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FORUM

Neuropsychology of Sports-Related Head Injury: Dementia Pugilistica to Post Concussion Syndrome*

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ABSTRACT

This article reviews the existing literature in the following areas of sports neuropsychology: Dementia Pugilistica, concussion and Post Concussion Syndrome, Second Impact Syndrome, and the emerging role of the sports neuropsychologist regarding return to play decisions. Dementia Pugilistica is discussed as a condition that exists along a continuum: Although many boxers will develop mild neurocognitive deficits, it is not yet known what percent of these mild presentations will progress to diagnosable Dementia Pugilistica. Factors contributing to both increased and reduced risk are detailed. The role of neuropsychological assessment in research and clinical management is reviewed. Existing studies of concussion incurred during contact sports provide evidence of an important role for neuropsychology in assessment and management of mild head injuries. Issues in clinical assessment of concussion are reviewed. The importance of grading of concussions, monitoring of postconcussive symptom resolution, and the use of neuropsychological test results in return to play decisions is detailed. The Second Impact Syndrome is discussed with regard to return to play decisions. Recommendations are proposed for research and for clinical application of findings in sports neuropsychology.

BACKGROUND

The neuropsychology of sports-related head injury is a new and developing field characterized by the diagnosis and treatment of the cognitive and emotional sequelae secondary to central nervous system injuries resulting from sporting activities. Neuropsychologists are increasingly examining and treating athletes sustaining head injuries ranging from the frequent uncomplicated mild concussion to the less frequent cerebrovascular compromise, edema, and Dementia Pugilistica. Athletes sustain these injuries in activities ranging from contact sports such as boxing and martial arts – in which contact to the head is integral – to football, rugby, hockey, and lacrosse – in which contact to the head is incidental – to non-contact sports not typically associated with head injury such as soccer, baseball, and basketball.

Sports-related head injuries represent approximately 20% of the 1.54 million head injuries estimated to occur annually in the United States. Of the total number requiring hospitalization, however, sporting injuries represent only about 9%, indicating that they are typically mild. For children under 15 years of age, sports accidents are the leading cause of medically attended head injury, exceeding motor vehicle accidents more than twofold. Among 15-to-24-year-olds,

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sports-related head injuries increase in incidence but are second in frequency to MVAs. Over the age of 25, sports-related head injuries decline sharply (Sosin, Sniezek, & Thurman, 1996).

Recent media attention has increased public awareness of sports-related head injuries. Both *Sports Illustrated* and ESPN have featured neuropsychologists in their work regarding concussion and their role in return to competition decision-making. Recent features in *USA Today* and *The New York Times* have also discussed these topics. Regarding severe injuries, millions of television viewers learned of Mohammed Ali's ''Parkinsonian Syndrome'' (thought to be a function of multiple concussions) during his dramatic appearance in the torch lighting ceremony of the 1996 Atlanta Summer Olympic Games.

This article reviews the existing research and clinical literature in the specialized field of sports neuropsychology: Dementia Pugilistica, mild concussion and Post Concussion Syndrome, Second Impact Syndrome, and the emerging role of the sports neuropsychologist in the clinical setting regarding return to play decision-making.

DEMENTIA PUGILISTICA

Background to the ''Punch Drunk'' Syndrome

The cognitive assessment of athletes sustaining head injuries initially focused on boxers. Martland (1928) was the first to describe the progressive syndrome variously referred to as ''Punch Drunk'', ''Dementia Pugilistica'' (Lampert & Hardman, 1984), ''Chronic Boxer's Encephalopathy'' (Serel & Jaros, 1962), ''Traumatic Boxer's Encephalopathy'' (Mawdsley & Ferguson, 1963) and a subtype of Chronic Traumatic Brain Injury or CTBI (Jordan, 1993). These authors depict the syndrome as manifesting early symptoms of mild confusion and ataxia and progressing to a ''Parkinsonian'' pattern of cognitive decline, impaired performance on memory tests, increased motor and speech latencies, dysarthria, pyramidal tract dysfunction, tremor in the head and upper extremities, and behavioral changes.

Neuropathological characteristics of Dementia Pugilistica (DP) includes cerebral atrophy, loss of cells in the cerebellum, and prominent neurofibrillary tangles in both cortical and subcortical areas, particularly in limbic structures and the substantia nigra (Corsellis, Bruton, & Freeman-Browne, 1973). Reduced cholinergic activity in the basal forebrain (Mendez, 1995) as well as β-amyloid plaques distributed diffusely and (for a subgroup) within vessel walls are characteristic of this syndrome as well (Jordan et al., 1995). A recent study has identified apolipoprotein E ε4 (APOE ε4) as a potential genetic risk factor for developing DP and chronic neurologic deficits during the course of a career in boxing and the resultant multiple mild head injuries (Jordan et al., 1997).

Studies indicate that DP results from numerous subconcussive and/or concussive blows to the head (Casson et al., 1984; Kaste et al., 1982). Several principle mechanisms of injury have been identified. Linear acceleration forces applied parallel to the surface of the head may result in axons being subjected to compressive and tensile stress (Cantu, 1996). Cerebrospinal fluid acts to mitigate the effects of compressive and tensile stress by absorbing and uniformly distributing the force about the brain. Nevertheless, focal ischemic lesions may result. Shearing stress occurs from a rotational acceleration force applied to the brain. Damage is characterized by diffuse axonal injuries, intracerebral and extracerebral hemorrhages, and edema (Lampert & Hardman, 1984). Also, carotid artery injuries may cause reflex hypotension with an associated brief dizziness. This can be especially dangerous for boxers, because the acceleration imparted by a blow will be greater if the athlete's neck muscles are relaxed (Cantu, 1996). Finally, a boxer may hit his head on the canvas, which may result in occipital coup and frontotemporal contracoup injuries (Lampert & Hardman, 1984).

Neuropsychological Investigations of DP

Studies on neuropsychological assessment of boxers have been subject to a number of limita-

tions inherent in the research designs: In some, appropriate control groups were not employed, or baseline data were not obtained and utilized for ipsative comparison. In others, participants were volunteers, raising the possibility of selection bias.

Early investigations of DP supported an etiology of multiple subconcussive blows (Casson, Sham, Campbell, Tarlau, & Didomenico, 1982) as well as cumulative effects of multiple concussions (Kaste et al., 1982). In the former study, 10 professional boxers underwent neurological, EEG, and CT examination shortly after being knocked out. Five had abnormalities on at least one of these exams. Four of these boxers had engaged in more than 20 bouts and none had been knocked out more than twice. The authors therefore attributed the abnormal findings to multiple subconcussive blows (Casson et al., 1982). Kaste et al. (1982) examined 14 amateur and professional boxers with CT and neuropsychological examination. All boxers were impaired on neuropsychological tests with two of the professionals demonstrating more severe impairment than the other boxers. Also, although 50% (3/6) of the professionals had abnormal CT findings, this was true for only 12% (1/8) of the amateurs. The authors concluded that the effects of repeated concussions are cumulative and ultimately cause irreversible brain damage. They also observed that their findings were consistent with clinical manifestation of DP as a slowly progressive condition. Along these lines, Sironi, Scotti, Ravagnati, Franzini, and Marossero (1982) found a relationship between abnormal CT and EEG findings and number of knockouts. However, in this study, length of career did not correlate with these findings.

As recently as 15 years ago, DP was perceived as a relatively rare condition that affected only vulnerable boxers who were either poorly trained, had little education, or had a history of substance abuse. The findings noted above were attributed to selection bias and lack of proper controls (Council on Scientific Affairs, 1983). Casson et al. (1984) therefore examined 18 boxers (15 former and active professionals and 3 amateurs), all of whom had no history of neurological illness and ‘‘responsible jobs, secondary or college educations, and no history of substance abuse’’ (p. 2663). Participants underwent neurological examination, EEG, CT, and neuropsychological testing. Of the 15 professionals, 13 had abnormal findings on at least two of the tests. The remaining professionals and the amateurs were impaired on at least one pair of the neuropsychological tests – immediate and delayed verbal memory. The number of concussions and amnestic episodes in this group was small and not related to impairment: Of 9 boxers with no history of concussion, 7 had abnormal findings on two or more tests. An impairment index, a composite of the multimethod findings, supported a direct relationship between length of professional boxing career and the presence and severity of neurologic damage. Neuropsychological testing was identified as the procedure most sensitive to cerebral dysfunction. Later studies (Butler, 1994; Haglund & Eriksson, 1993) have provided additional evidence that low exposure variables such as amateur status and low number of bouts are associated with fewer significant findings of head injury.

In an effort to gain a better understanding of the early effects of multiple subconcussive blows, McLatchie et al. (1987) compared 20 amateur boxers to an equal number of orthopedic outpatients with limb fractures. Among the boxers, one had an abnormal CT, 7 had abnormal clinical examinations, and 8 had abnormal EEG findings. Boxers’ impairments on neuropsychological tests were significant relative to controls. EEG was thought to be inconclusive evidence of cerebral dysfunction and the abnormal clinical findings were perceived as minor and insufficient to diagnose brain disease, especially without the benefit of serial examination. Neuropsychological measures of verbal and visual memory, attention, and reaction time were therefore deemed the most sensitive measure of the presence of neurologic dysfunction. The authors noted however, that while many of the boxers scored within normal limits on many measures, a few demonstrated severe impairment. These impaired performances were largely responsible for the significant differences between the boxers and the control group. Thus, even in young amateurs, some boxers ap-

peared more vulnerable than others to neuropsychological impairments. This may help to explain the results of two similar studies of young boxers and matched controls (Brooks 1987; Levin et al., 1987) in which no significant deficits on neuropsychological measures were found.

A more recent study of 50 amateur boxers by Haglund and Eriksson (1993) detected mild EEG deviations and inferior fine motor speed compared to control groups consisting of soccer players and field athletes. Also, Stewart et al.'s (1994) two-year prospective study of amateur boxers found significant trends between number of bouts prior to baseline assessment and changes in memory and visuomotor constructional ability; however, no significant changes were detected over the longitudinal course of the study. The authors hypothesized that this may have reflected a long latency for effects to become manifest and/or prior changes in safety practices that created a selection bias.

In a recent intriguing study, Jordan et al. (1997) compared cognitive-mental status test performances of 18 high exposure (12 or more professional bouts) and 12 low exposure boxers with known APOE genotypes. APOE (apolipoprotein E) has been identified as a susceptibility gene for late onset familial and sporadic Alzheimer disease (AD) (Strittmatter et al., 1993). Located on chromosome 19q13.2, it occurs in three allelic forms designated ε2, ε3, and ε4. Pairings give rise to six possible genotype combinations. One copy of APOE ε4 is associated with a moderately increased risk of AD, and two copies are associated with a more significant risk and an earlier age of onset (Corder et al., 1993), although the precise risk-enhancing factors remain to be identified (Martinez et al., 1998). APOE ε2 may serve a neuroprotective function as it is underrepresented in AD populations (Corder et al., 1994). Pathologically, AD and DP are similar in regard to the presence of neurofibrillary tangles, diffuse β-amyloid plaques, and reduced cholinergic activity in the basal forebrain (Hof et al., 1992; Uhl et al., 1982). Noting that head trauma has been identified as a potential environmental trigger for AD onset (Van Duijn, 1996), the authors hypothesized that boxers' APOE genotype status may indicate an increased risk of DP and its severity among boxers.

The boxers' genotype distribution was similar to that of the general population. Of the boxers with severe DP, all (100%) had at least one copy of the APOE ε4 allele. Of those diagnosed in the moderate DP range, 50% had the allele. Only 25% of mild DP boxers and 18% of those with normal examinations had one or more copies. This finding was statistically significant ($p < .01$). Furthermore, the presence of the APOE ε4 allele in high exposure boxers was found to be significantly related ($p < .001$) to the presence of more severe DP symptoms. The authors concluded that the APOE ε4 allele is associated with an increased risk for DP in high exposure boxers. Due to the small sample size, they were unable to conclude definitively whether the allele is associated with symptoms in low exposure boxers. The authors also reported a possible protective factor due to the presence of an APOE ε2 allele in high exposure boxers with few indicators of DP. Finally, the authors noted that their finding of moderate to severe encephalopathy in 23% of their participants is roughly comparable to other estimates (e.g., 17% in Roberts, 1969) regarding prevalence of DP. The theoretical links and hypothetical association of mechanisms of action of DP with AD supported by this study merit serious consideration and further research. However, replication of Jordan et al.'s findings with larger sample sizes are necessary before drawing definitive conclusions regarding the role of APOE in DP.

Neuropsychological assessment has already played a key role in research on DP since it is the most sensitive method for early detection of symptoms. Serial testing of boxers' cognitive functioning for early signs of neurologic dysfunction is useful for amateur and professional athletes – especially those with significant risk factors – in making important decisions about future health and career issues.

Summary

Dementia Pugilistica is a condition that exists along a continuum. Although many boxers will develop mild neurocognitive deficits early in

their career, it is not yet known what percent of these mild symptom presentations will progress to diagnosable DP. Prevalence of moderate to severe cases of DP is approximately 20%, and increased risk is associated with exposure variables and number of concussions. Both increased and reduced risk appear to be associated with genetic factors. Neuropsychological assessment is critical for early detection of DP and is likely to remain an important tool for research. Clinical use of neuropsychological test data in advising amateur and professional boxers regarding health and career issues should be considered, particularly for those with known DP risk factors.

CONCUSSION AND POST CONCUSSION SYNDROME

Athletes and Concussion

In contrast to boxing, in which the symptoms of interest are primarily the result of multiple subconcussive blows, neuropsychological investigations of football players and other athletes who engage in contact sports such as rugby, lacrosse, and hockey are typically concerned with the immediate and ongoing effects of individual and multiple concussions. Estimates for football head injury alone in the US range from 100,000 to 250,000 per year (Cantu, 1988; 1996). Barth et al. (1989) reported that 10% of all college football players will sustain a mild head injury over a given season and that 40% will do so over the extent of their high school and college careers. Indeed, neuropsychological assessment of concussion is becoming widely accepted in several professional and college sports venues. The National Football League has established a subcommittee for the study of concussion and Post Concussion Syndrome and approximately half of the franchises currently utilize neuropsychological assessment as a component in their return-to-play decision making process. Also, a league-wide assessment and research protocol is currently being utilized in the National Hockey League (Lovell, 1997).

Because of its frequency in non-sports venues, concussion has been the subject of many clinical investigations. It is typically defined as a traumatically induced alteration in mental status with or without loss of consciousness (duration less than 20 min if positive LOC), a Glasgow Coma Scale score ranging from 13-15, less than 20 min loss of consciousness, and negative findings on neuroimaging (Rimel, Giordani, Barth, Boll, & Jane, 1981). Concussions are thus considered to be a type of mild head injury. Symptoms include headache, dizziness, diplopia, nausea, fatigue, along with cognitive weaknesses (Alexander, 1995). Neurocognitive deficits occur in areas of information processing, attention/concentration, reasoning, visuospatial processing, and memory (Katz & DeLuca, 1992). Typically, ongoing symptoms – known as Post Concussion Syndrome (PCS) – resolve in a relatively short period of time, although cognitive deficits and difficulty returning to work have been noted 3 months posttrauma (Rimel et al., 1981). In a multicenter study, Ruff, Levin et al. (1989) found memory dysfunction to be nearly resolved at 1 month. A study by Dikmen, McClean, and Terrkin. (1986) found significant recovery relative to controls at 3 months and near equivalence to a control group at 1 year. Nevertheless, a small number of patients appeared to have symptoms that persisted more than one year.

Pathophysiological changes in concussion are set in motion when a blow to the head causes acceleration of the brain, resulting in axonal strain (Gennarelli, 1986). Long tract axons are stretched or compressed causing alterations in axon integrity and/or permeability, in isolation from focally related brain parenchymal or intraparenchymal vascular abnormalities (Polivshock & Coburn, 1989). Such multifocal axonal injuries have been identified in postmortem examination of patients who sustained concussions entailing as little as 60 s of loss of consciousness and who subsequently died of other causes (Blumbergs et al., 1994). Important parallels to the sports domain have been established through the identification of diffuse axonal injury fol-

lowing assault (Graham, Clark, Adams, & Gennarelli, 1992; Imajo, Challener, & Roessman, 1987) and fall (Abou-Hamden et al., 1997). (For a review of diffuse axonal injury, see Gennarelli, Thibault, & Graham, 1998).

Recent experimental models of mild head injury in animals have provided further information on physiological effects of concussion. Immediately following fluid percussive head injury, there is an increase in extracellular potassium of the rat hippocampus. The influx of potassium appears to be secondary to sudden intense neuronal discharges (Katayama, Becker, Tamura, & Hovda, 1990). Although not mechanically damaged from impact, neuronal cells exposed to concussive injury dramatically altered their metabolic functioning. It is the induced ionic flux that most likely alters the cellular energy, and there is evidence that the prolonged accumulation of calcium (Ca^{2+}) contributes to this injury-induced ionic flux (Fineman, Hovda, Smith, Yoshino, & Becker, 1993). Concussed rats demonstrated bilateral hypermetabolism immediately following injury. The hypermetabolism was followed as early as 6 hr postinjury by a period of metabolic depression which lasted for as long as 5 days (Yoshino, Hovda, Kawamato, Katayama, & Becker, 1991).

Cerebral hyperglycolysis, an increase in glucose utilization, has been shown to be a pathophysiological response to head injury-induced ionic and neurochemical cascades. Here, concussive events lead to an abnormal cellular state of increased glucose utilization. An increased glycolytic generation of adenosine triphosphate may indicate a compensatory response to avert failure. Under normal conditions increases in glucose metabolism are matched with appropriate increase in cerebral blood flow. Therefore, concussion is seen to produce a state of relative ischemia with regards to the metabolic demand of the tissue (Bergsneider et al., 1997).

Although concussions sustained in contact sports are similar to those occurring in non-sports contexts, they differ in one significant aspect. Specifically, sports-related concussions are typically the result of a relatively low velocity impact compared to that resulting from the acceleration/deceleration forces set in motion during a motor vehicle accident, the most common immediate cause of concussion in clinical settings. Rotation and shear strain is therefore less severe and the majority of sports-related concussions fall in the mild range of severity. Episodes of confusion and disorientation are more common than the loss of consciousness associated with more severe concussion. (Athletes refer to these events informally as ‘‘Dings’’ and having one’s ‘‘bell rung.’’) However, as in boxing, if a blow is not anticipated – as when a player is tackled from behind – acceleration forces to the head can be significantly increased (Cantu, 1996).

Assessment of Severity of Concussion

Much of the sports literature deals with the assessment of concussion severity and the determination of fitness for return to play. In football, assessment of severity has been accomplished by use of a graded hierarchy of neurobehavioral patterns. Torg (1982) originally outlined a four-grade paradigm. The mildest grade of concussion was defined as confusion without amnesia (Grade 1). Intermediate stages were characterized by the presence of posttraumatic amnesia (Grade 2) and both posttraumatic amnesia and retrograde amnesia (Grade 3). The most severe category (Grade 4) entailed loss of consciousness. The most recent paradigm, a Practice Parameter issued by the American Academy of Neurology in 1997, establishes three grades of concussions and the associated guidelines for return to competition in contact sports (Practice Parameter, 1997). A Grade 1 concussion (mild) is characterized by transient confusion, no loss of consciousness, and concussion symptoms or mental status abnormalities on examination that resolve in less than 15 min. Athletes with Grade 2 concussions (moderate) manifest transient confusion, no loss of consciousness, and concussion symptoms or mental status abnormalities on examination that last more than 15 min. Grade 3 concussions (severe) are those that entail loss of consciousness.

Other grading classification systems and return to play guidelines have been developed in previous years (Cantu, 1991; Colorado Medical Society, 1991; Jordan, 1994). In outlining his

four-grade scale, Jordan (1994) makes a strong case for distinguishing between brief and prolonged LOC in the classification of severity, and Cantu (1991) suggests attention to the issue of posttraumatic amnesia (PTA). These and other grading systems are included in the Appendix.

Cantu (1996) has pointed out the need for standardization of assessment as a prerequisite for meaningful large-scale studies. Two recent efforts have attempted to address this problem. In their norm-referenced approach, McCrea and colleagues identified the domains of orientation, immediate and delayed memory, and concentration as pertinent to the assessment of concussion. Utilizing a sample of high school football players, they then obtained baseline rates for individual players as well as group norms. In the initial six trials, their sideline assessment measure (Standardized Assessment of Concussion, SAC) was found to be sensitive to the immediate effects of mild concussion using both ipsative and group norms (McCrea, Kelly, Kluge, Ackley, & Randolph, 1997). In a larger sample of 33 high school and college players, the instrument distinguished concussed from non-concussed players and was useful in tracking resolution of mental status abnormalities (McCrea et al., 1998). The authors suggest that the instrument may prove useful in determining fitness for return to play, particularly when symptoms do not persist.

Taking a standardized approach to the assessment of concussion, Kutner et al., (1998) have developed the Sideline Concussion Checklist (SCC). The SCC was developed to provide a standardized format for evaluating a player's ability to return to play in the same game. This instrument tracks the resolution over time of physiological, neurological, and cognitive symptoms associated with the immediate effects of concussion. It is therefore useful in systematically classifying concussions according to the grading system established by the American Academy of Neurology. Also, the identification of specific symptoms on the SCC allows for comparison of concussions at various sites with different raters, making large scale studies feasible. Finally, (in keeping with Yarnell & Lynch, 1973), the SCC utilizes assessment strategies immediately relevant to the situation. For example, players are asked about a previous play or about specific play assignments to assess retrograde and remote memory functioning. This instrument is currently used by several National Football League (NFL) teams.

Assessment of Post Concussion Symptoms and Return to Competition

Decision-making regarding an athlete's return to competition is the critical issue. Ideally, athletes who are fully recovered and not at risk should be not be prevented from continued participation. A number of guidelines have been published, including the 1997 Practice Parameter and those of Cantu, Jordan, and the Colorado Medical Society (Appendix). These formats are not fully researched, but rely on professional judgement. Generally speaking, all the formats are similar. It is recommended that the return-to-play decision include use of one of these formats.

Neuropsychological assessment offers many advantages over standard neurological examination and other radiological and neurophysiological procedures for the evaluation of postconcussive symptoms and decisions regarding return to play. Following resolution of the immediate effects of concussion (e.g., disorientation, ataxia, diplopia, nausea, etc.), athletes may continue to manifest a pattern of physiological, affective, and cognitive symptoms (Levin, 1994). As outlined in the Appendix, it is recommended that players be asymptomatic for a period of time (typically 1 to 2 weeks) before being allowed to return to competition. At this stage, electroencephalogram (EEG) tracings and brain stem auditory-evoked responses (BAER) studies are not consistently useful for the diagnosis of postconcussive symptoms (Jacobson & Sperling, 1993; Schoenhuber & Gentilini, 1989). Quantified EEG (QEEG) is a research tool only and cannot be used to diagnose mild head injury (Jacobson & Sperling, 1993). Neuroradiological studies are useful in ruling out infrequent but more serious sequelae of concussions such as contusions, intraparenchymal pathology and edema. However, they have not been shown to be sensitive to the effects of mild head injury (Bigler & Snyder, 1995; Eisenberg & Levin,

1989; Rimel, Giordani, & Barth, 1982). PET and SPECT scans remain research tools at this time (Humayun, Presty, & Lafrance, 1989; ''Therapeutic,'' 1996). Self-report measures of symptoms have inherent weaknesses well known to the neuropsychologist (Lezak, 1995). While useful in assessing the effects of a moderate or severe concussion, the traditional Mental Status Exam is not sensitive to subtle attentional and memory weaknesses and does not allow for adequate documentation of improved performance over time (Ruff, Levin, & Marshall, 1986). Neuropsychological tests are therefore becoming an important factor in decision-making regarding return to competition (Kutner, Warren, & Barnes, 1997).

The first major study to establish the utility of neuropsychological testing in this sports context was Barth and colleagues multicenter investigation (Barth et al., 1989; Macciocchi, Barth, Alves, Rimel, & Jane, 1996). In this four-year prospective study, preseason baseline data were gathered on 2,350 college football players on measures of attention/concentration (Trail Making Test, TMT; Reitan & Davison, 1974), psychomotor problem solving/visual perceptual ability (Symbol Digit Modality Test, SDMT; Smith, 1973), and complex sustained attention/immediate recall memory and rapid mental processing (Paced Auditory Serial Addition Task, PASAT; Gronwall & Sampson, 1974; Gronwall & Wrightson, 1980). Players who sustained a concussion were retested at 24 hr postinjury, 5 days postinjury, 10 days postinjury, and postseason. To control for practice effects and affective sequelae of injury, similar data were collected on athletes who sustained mild orthopedic injuries and normal controls (students). Statistical analysis revealed concussed players did not demonstrate the expected practice effect on the SDMT and PASAT measures. Consistent with the observation of Strauss and Allred (1987), the authors interpreted this as evidence of cognitive impairment. Moreover, the concussed players' scores on these measures improved over the ten day retest period and then leveled off. The authors concluded that neuropsychological tests were sensitive to the effects of a single mild concussion 24 hr following injury and that significant recovery takes place for most athletes within a 5 to 10-day period. They hypothesized that an information processing deficit might underlie the concussed players' defective performances.

Additional insight in this area has been obtained from studies of Australian Football League players carried out by Maddocks and Saling (1991, 1996). Noting that immediate neurological symptoms such as headache, nausea and vertigo might not have resolved within 24 hr and could therefore affect concussed players' performances on neuropsychological measures, assessments in their studies were carried out at 5 days postinjury. For an initial 10 participants, the authors reported reduced performances on the Digit Symbol Substitution Test (DSST; Wechsler, 1981) and Four Choice Reaction Time – decision time (Van Zomeren, 1981). The PASAT was also administered but was not found to be sensitive, as it had been in the Barth et al. (1989) study. Differences in stimulus presentation rates and/or populations across studies were hypothesized to account for this result. The authors concluded that neuropsychological deficits are detectable in the early stages following mild concussive injury following resolution of neurological and neurobehavioral symptoms.

A recent study of concussive injuries in professional rugby players (Hinton-Bayre, Geffen, & McFarland, 1997) followed up on Barth et al.'s (1989) hypothesis that an information processing deficit might underlie impaired performances on neuropsychological tests by concussed athletes. Drawing from a representative sample of rugby players, the authors first examined the effects of repeated testing for measures hypothesized to be sensitive to the effects of concussion (i.e., tests of information processing) and on ones hypothesized to be resistant to those effects (i.e., tests of premorbid ability). Ten concussed players were subsequently re-examined on these measures postinjury and performed as hypothesized. Findings confirmed the utility of two timed tests requiring speeded information processing (SDMT and DSST) that had been previously found to be sensitive to the effects of concussive injury in athletes. A third measure, the Silly Sentences subtest of the

Speed and Capacity of Language Processing Test (SCOLP; Baddeley, Emslie, & Nimmo-Smith, 1992) was found to be the most sensitive. However, the authors found that for the SDMT, postinjury deficits were more evident when compared to an initial baseline score rather than a retest baseline. They therefore suggested that multiple baselines be obtained so that comparisons can be made to an optimal preinjury level of performance.

Indeed, Bleiberg, Halpern, Reeves, and Daniel (1998) found in a research setting that cognitive sequelae of concussion may manifest as intraindividual inconsistency over multiple testings. In a computerized assessment of reaction time, concussed patients and controls were administered a customized battery 30 times across 4 days. Controls each manifested clear learning curves. Concussed individuals demonstrated variable performance over the 4 days with some appearing more impaired on days 3 and 4 than on days 1 and 2. The authors suggest that the ability to perform cognitive measures with stability across days may be the important indicator of recovery from concussion.

Lovell and Collins (1998) have gathered pre- and post-season test-retest data on 40 college football players for a similar battery: Hopkins Verbal Learning Test (Brandt, 1991), Trail Making, Digit Span, Symbol Digit Modality Test, Grooved Pegboard, and COWAT (Benton & Hamsher, 1978). Minimal but significant practice effects were identified on the Trail Making Test, Part A and COWAT, demonstrating the stability of the battery over longer time periods for athletes who may undergo multiple baseline assessments.

Thus far, clinical reports of neuropsychological test data's use as a component of return to play decision-making are few. In professional sports, Kutner, Warren, and Barnes (1997) provided details of the protocol utilized in the NFL by the New York Giants. Baseline levels of information processing speed and information processing accuracy were obtained on athletes by means of a computerized administration of MicroCog (Powell et al., 1993). The authors note several advantages of this testing medium, including ease and standardization of administration and precise quantification of reaction time. Two of three concussed athletes were re-examined 5 days postinjury and found to have no neurocognitive deficits. Both returned to competition for the following game. One player was found to have neurocognitive deficits following a concussion and remained out for the following game. This player was re-examined 10 days postinjury and found to have returned to baseline status. He subsequently returned to competition for the next game. Neurocognitive data were interpreted by a board-certified clinical neuropsychologist and were utilized along with team physician examination and trainer consultation for decisions regarding return to competition.

Lovell and Collins (1998) have reported their findings in four cases of college football players who were diagnosed with concussion and tested within 24 hr. Relative to baseline testing, all four manifested declines on the Symbol Digit Modalities Test, three demonstrated a drop on COWAT, and two a decline on the Trail Making Test, Part B. The authors observed that their findings are consistent with deficits in information processing speed and verbal fluency, but not motor speed/coordination or verbal memory. Noting that none of the concussed athletes experienced loss of consciousness, the authors pointed out both the sensitivity of the measures and the importance of neuropsychological testing in the detection of lingering deficits and formulation of return-to-play recommendations.

Guskiewicz and Perrin (1995) reported use of the PASAT in conjunction with a postural sway test for case management of a 17-year-old high school football player who received a mild concussion. Baseline PASAT performance levels had been obtained during the preseason. Following injury, the athlete was re-examined on days 1, 3, 5 and 10. Both postural sway and PASAT findings were abnormal compared to baseline on day one postinjury. By day 3, postural sway, but not PASAT, had returned to near preseason levels. PASAT data revealed increases from day 1 through day 5 and then leveled off. This is consistent with Maddocks and Saling's (1996) finding that neuropsychological deficits are detectable after resolution of neurological symptoms

in the early stages of a mild concussive injury. The athlete was allowed to return to practice with no contact after testing within normal limits on day 5. He participated in a full practice on day 6, and on day 7 returned to play on a limited basis.

Post Concussion Syndrome

The findings of Barth et al., (1989) suggest that complete or near complete recovery from mild concussions incurred by most football players is reached within 10 days. However, in the less frequent severe cases there is sufficient reason for concern regarding the development of Post Concussion Syndrome (PCS). Several investigations of non-sports concussion have documented chronic PCS in individuals sustaining mild head injuries (Barth et al., 1983; Rimel, Giordani, Barth, Boll, & Jane, 1981; Silverbåge Carlsson, Svårdsudd, & Welin, 1987). Ruff et al. (1994) found corroborative evidence of poor long-term outcome following mild head injury as detected by neuropsychological testing, utilizing positron emission tomography (PET) studies regardless of whether the concussion entailed loss of consciousness.

Moreover, anecdotal evidence of PCS in athletes is common, particularly following multiple concussions (e.g., Farber, 1994). The cumulative effects of repeated concussive injuries among non-athletes have been well documented (Gronwall & Wrightson, 1975; Silverbåge, Carlsson, Svårdsudd, & Welin, 1987), and there is significant evidence of multiple concussive events among athletes. Barth et al. (1989) found that 42% of college football players reported a history of concussion prior to the beginning of their study, 11% reported a history of two concussions, and 11.6% reported a history of three or more. Separate studies have estimated that players who sustain one concussion are four to six times more likely to sustain a subsequent concussion (Gerberich, Priest, Boen, Straub, & Maxwell, 1983; Zemper, 1994). Although no evidence establishing a genetic predisposition to chronic PCS has yet been identified, Jordan et al.'s (1997) previously cited works may be relevant to this area of study.

Chronic PCS has been investigated in studies of active and retired professional soccer players. Although not considered a traditional contact sport, the effects of years of ‘‘heading’’ soccer balls has generated concern. A number of studies have demonstrated EEG abnormalities, unexpected cerebral atrophy, chronic PCS, and significant neurocognitive deficits in older and more experienced soccer players which may relate to body to body contact and perhaps heading of the ball (Abreau, Temple, Schuyler, & Hutchison, 1990; Tysvaer & Lochen, 1991; Tysvaer, Storli, & Bachen, 1989; Witol, 1995). One recent neuroradiological study of player physiological complaints, however, found PCS symptoms to be correlated with player histories of prior acute head injury, not length of career or number of headers (Jordan, Green, Galanty, Mandelbaum & Jabour, 1996).

Finally, it must be noted that although some athletes sustain mild head injuries and manifest transient and/or persisting cognitive deficits, there are cases in which concussed individuals do not appear to manifest cognitive sequelae. This suggests that differences in resilience and individual recovery factors may exist and that possible psychogenic and motivational factors may play a role in persisting postconcussive cognitive symptoms in some cases. (See Binder, Rohling, & Larrabee, 1997 and Binder, 1997 for a meta-analysis and review of factors influencing persisting cognitive sequelae of mild head injury).

Summary

This review of the literature on concussions incurred during contact sports provides evidence of an important role for neuropsychology in the assessment and management of sports-related mild head injuries. First, sports-related concussions – although by definition within the diagnosis of mild head injury – can be graded according to relative level of severity. Standardized neurobehavioral-cognitive assessment of the immediate effects of these injuries is useful for both clinical case management and research purposes. Second, neurocognitive deficits are detectable after resolution of neurological symptoms. Neuropsychological testing should therefore play an important role in return to play de-

cision-making. Individual baseline scores are necessary and multiple baseline scores may be indicated for accurate interpretation of post-injury test data. Assessment measures that have been found to be sensitive to the effects of multiple concussion are those measuring information processing speed and efficiency. These tests are also useful for tracking rate and extent of neurobehavioral recovery. Chronic Post Concussion Syndrome may affect selected athletes, particularly following more severe and multiple concussions.

SECOND IMPACT SYNDROME

While the chronic effects of a single or multiple concussions are of concern, it is the catastrophic effects of the Second Impact Syndrome (SIS; Saunders & Harbaugh, 1984) that require careful monitoring of the resolution of concussive symptoms. Unfortunately, little is understood about possible individual predisposition or symptom patterns related to this syndrome.

SIS occurs when an athlete who sustains a head injury subsequently receives a second head injury before full resolution of the first symptoms. Re-injury need not occur on the same day (‘‘Sports-related,’’ 1997) and has been reported up to 4 days following first concussion with brief loss of consciousness (Saunders & Harbaugh, 1984). The second impact may appear to be minor, with the athlete appearing slightly dazed and walking off the field without assistance. Shortly thereafter the athlete may collapse and lose consciousness with rapidly dilating pupils. Respiratory failure ensues and death may occur (Cantu, 1992). The immediate cause of death is typically brain swelling, increased intracranial pressure and subsequent herniation of either the uncus of the temporal lobe or of the cerebellar tonsils through the foramen magnum causing brain stem failure. The apparent physiological mechanism is failure of the autoregulation of cerebral circulation, causing vascular engorgement (Kelly et al., 1991).

There appears to be growing support for a ‘‘critical period’’ for optimal recovery from a concussive event. The period is related to the cerebral maturation of the brain, rather than age as determined by birth. Evidence for this again comes from the animal literature. In a seminal project by Prins, Lee, Cheng, Becker, and Hovda (1996), fluid percussion head injury was evaluated in developing and adult rats. Compared to adult animals, developing rats exhibited pronounced hypotension in response to closed head injury. These researchers noted that this finding may explain the mass edema cases which are evident in children, and it is also to be noted that a review of the Second Impact Syndrome reveals no cases in which the individual is more than 21 years of age. Although this may only reflect sports participation figures, there may be more risk of SIS in developing versus adult brains.

It is to be noted that the entity of SIS has been recently questioned. McCrory and Berkovic (1998) reviewed the literature for SIS cases. Of the cases reviewed they identified 17 as being consistent with SIS. Formulating a set of four criteria, they found that none of the cases met all the criteria, but that five cases met three criteria. The concluded that SIS does not exist or that it is overdiagnosed.

Clearly, SIS is a rare and little studied condition. However, two critical factors need to be considered. First, SIS is characterized by severe morbidity. Second, there are no published studies indicating that this condition does not exist. Taken together, these factors should direct the neuropsychologist to consider SIS as a real entity at this time.

Neuropsychology’s role regarding SIS is still being defined. Regarding return to play decision-making, as noted above, neuropsychological tests have been found to be quite sensitive for monitoring PCS cognitive symptoms. Moreover, the above findings suggest that younger players (≤ 21) may be at high risk for SIS, thus it is critical that they not be allowed to return to play until they are asymptomatic. Finally, standardized neuropsychological assessment of the immediate effects of concussion may prove useful along with other research tools in identifying hallmark symptom patterns and predisposing risk factors.

RECOMMENDATIONS

The above findings suggest several directions for future research as well as opportunities for clinical application of current findings in the field of sports neuropsychology.

1. Longitudinal or cross-sectional investigation of boxers could help identify additional factors affecting the course of DP. Such knowledge could aid in genetic counseling of boxers who develop mild neurocognitve deficits and have known risk factors.

2. Baseline neuropsychological testing of active boxers and other athletes would allow for tracking of postconcussion resolution of symptoms before a return to competition.

3. Standardized sideline assessment of concussion in contact sports is necessary for gaining a better understanding of whether there are risk factors associated with Post Concussion Syndrome. Neuropsychological test data should become a standard component of return to play decision-making in contact sports.

4. Genetic testing in conjunction with neuropsychological assessment may be useful in exploring the relationship of chronic PCS and DP.

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APPENDIX

Concussion Grades and Return To Competition Guidelines

Practice Parameter (1997) The management of concussion in sports (summary statement), *Neurology, 48*, 581-585.

Grade 1	Grade 2	Grade 3
1. Transient Confusion 2. No LOC 3. Concussion symptoms or mental status abnormalities on examination resolve in **less than 15 min**	1. Transient Confusion 2. No LOC 3. Concussion symptoms or mental status abnormalities on examination **last more than 15 min**	1. Any LOC, either brief (seconds) or prolonged (minutes)
May return to play if postconcussive symptoms resolve within 15 minutes. Any athlete who receives a second Grade 1 concussion on the same day should be removed from play until asymptomatic for 1 week.	Return to play if asymptomatic for 1 week. Any athlete who receives a Grade 2 concussion subsequent to a Grade 1 concussion on the same day should be removed from play until asymptomatic for 2 weeks.	Removed from play for 1 week without symptoms if LOC is brief or 2 weeks without symptoms if prolonged. An athlete who receives a second Grade 3 concussion should be removed from play until asymptomatic for 1 month. If CT, MRI or other findings, player should be removed for the season and discouraged from further participation in contact sports.

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Grade 1 *(Mild)*	Grade 2 *(Moderate)*	Grade 3 *(Severe)*
No LOC and PTA < 30 min	LOC < 5 min or PTA = 30 min to 24 hr	LOC > 5 min or PTA > 24 hr
May return to play if asymptomatic for 1 week.	May return to play if asymptomatic for 1 week.	Should not be allowed to play for at least 1 month. May then return to play if asymptomatic for 1 week.

Colorado Medical Society. (1991). *Report of the Sports Medicine Committee; Guidelines for the Management of Concussion in Sports (revised).* Denver: Colorado Medical Society.

Grade 1	Grade 2	Grade 3
Confusion without amnesia No LOC	Confusion with Amnesia No LOC	Any LOC
May return to play if asymptomatic at rest and exertion after at least 20 min	May return to play if asymptomatic for 1 week	Should not be allowed to play for at least 1 month. May then return to play if asymptomatic for 2 weeks

Jordan, B.D. (1994). *Sports injuries.* National Athletic Trainers' Association Research and Education Foundation. Proceedings, Mild Head injury in Sports Summit, Washington DC.

Grade 1 *Mild*	Grade 2 *Mild-Moderate*	Grade 3 *Moderate-Severe*	Grade 4 *Severe*
1. Confusion without Amnesia 2. No LOC	1. Confusion 2. Amnesia <24 hr 3. No LOC	LOC with Altered Level of Consciousness Not Exceeding 2–3 min	LOC with an Altered Level of Consciousness Exceeding 2–3 min
Return to play if asymptomatic at rest and after exertion following at least 20 min of observation	May return to play if asymptomatic for 1 week	Should not be allowed to play for at least 1 month. May then return to play if asymptomatic for 1 week	Should not be allowed to play for at least 1 month. May then return to play if asymptomatic for 2 weeks